AN INVESTIGATION INTO THE USE OF THE STANDARD RADIOGRAPHIC HAND IN THE CLASSIFICATION OF CHRONIC ARTHRITIS AND CERTAIN DISEASES ASSOCIATED WITH A DISTURBANCE OF CALCIUM METABOLISM.

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An investigation into the use of the STANDARD RADIOGRAPHIC HAND in the classification of chronic arthritis and certain diseases associated with a disturbance of Calcium Metabolism.

When we come to consider that there are no less than thirty or forty names or types of Rheumatic Arthritis described, any method of investigation which will help to enlighten this chaotic state of affairs, will be worthy of examination. Not only are we over-burdened with names but these names mean something different to each individual. The following is a list of names in common use:-

Nomenclature of Chronic Arthritis. (1)

1. Capsular.
2. Dry.
3. Adhesive.
4. Rarefying.
5. Villous.
6. Monarticular.
7. Polyarticular.
8. Specific infective.
9. Rheumatoid.
10. Chronic infectious.
11. Atrophic.
12. Deformans.
13. Rheumatic.
14. Chronic rheumatic.
15. Chronic rheumatism.
16. Primary progressive.
17. Secondary progressive.

18. Chronic proliferative.
19. Chondro-osseous.
20. Osteoarthritis.
21. Degenerative.
22. Hypertrophic.
23. Climacteric.
24. Menopausal.
25. Senile.
26. Chronic static.
27. Traumatic.
28. Metabolic.
29. Gouty.
30. Mixed.
31. Arthrosis.
32. Metastatic.

Dr. R. E. Gordon, a few years ago at the Royal Society of Medicine, made the following statement: "Osteo-arthritis is one of those Clinical entities with no very clear definition, no very clear pathology, no very clear limitations of clinical symptoms and no very clear treatment."

I hope to be able to show that this criticism can no longer hold good.

The importance of this subject of Arthritis and Rheumatic diseases can be appreciated from the report of the Ministry of Health published in 1926 : "nearly one-sixth of the industrial invalidity of this country is due to diseases classed as Rheumatic. Each year these diseases are costing nearly £2,000,000, in Sick Benefit, and over three million weeks of work are lost annually."

Strangeway's classical work on the pathology of Arthritis has been our mainstay in the past but it has

certain disadvantages when we come to consider nomenclature and classification.

Arthritis can be classified in four different ways:-

a) Aetiologically.
b) Clinically.
c) Pathologically.
d) Radiographically.

I propose in this Thesis to describe each of these groups and to demonstrate the use that can be made of a radiogram of the hand both from the point of view of accurate diagnosis and also its use in the estimation of bone calcium in various other diseases.

Since I began the present investigations, the Report of a special Committee, appointed by the Council of the British Medical Association, on the Causation and Treatment of Arthritis and Allied Conditions, has been published.

It has been of immense value in clarifying the present position; however, as I adopted the nomenclature and classification which are used at the British Red Cross Clinic at Marylebone when I began this work, I will adhere to it.

THE CLASSIFICATION OF ARTHRITIS AND ALLIED DISEASES.

That adopted by the British Medical Association (a).

is as follows:-

	Synonyms.	
Rheumatoid Arthritis.	Chronic poly-arthritis. (Continental nomenclature).	Primary: cause unknown; this as knowledge increases may merge into
	Atrophic (Goldthwait).	
	Proliferative (Nichols & Richardson). (American nomenclature).	Secondary: associated with focal or general infection.
Chronic Villous Arthritis		Mainly occurring in women at or about the climacteric.
Osteo-Arthritis.	Hypertrophic (Goldthwait)	Primary: no definite association with infection.
	Degenerative (Nichols & Richardson).	Secondary: associated with infection.
Spondylitis	Ankylopoietica	Arthritis of spinal joints, with bony ankylosis, spreading centrifrugally to adjacent large joints.
	Osteo-arthritica.	Osteo-arthritis of the spine. (The labourer's spine).
Fibrositis.	Intramuscular and fascial.	
	Periarticular.	
	Bursal and tenosynovial.	
	Subcutaneous (panniculitis).	
	Perineuritic.	

THE CLASSIFICATION OF RHEUMATIC DISEASES OF JOINTS AND TISSUES.

(in use at the British Red Cross Clinic, Marylebone.)

This classification is aetiological and aims, in the present state of our knowledge, at eventually abolishing anatomical distinctions.

CLASS A. ARTICULAR.

1. Infective. (a) Cause unknown. (non-specific)
   (b) Cause known. (specific- e.g. scarlet fever, gonococcal.)
2. Traumatic.
3. Degenerative. (a) Senile. (common chronic osteo-arthritis.)
   (b) Climacteric. (Villous type of B.M.A. classification.)
4. Metabolic. (a) Gout.
   (b) Rheumatoid arthritis.
   (c) Others. (True allergic conditions).
5. Miscellaneous e.g. neuropathic, scurvy, haemophilia, hysterical joint.

CLASS B. NON-ARTICULAR. To be grouped as far as possible, in the same way as the articular group.

CLASS C. NON-RHEUMATIC CONDITIONS.

I will now give a brief description of the chief clinical and aetiological features of the groups that are to be considered in this Thesis.

INFECTIVE ARTHRITIS.

The differentiation of the two clinical varieties of arthritis is usually straightforward. The actual joint lesion, on the other hand, is fundamentally the same except, of course, in the grossly suppurative case.

The main clinical features common to both varieties are as follows:-

1. Slow onset in the general disease.
2. Synovial effusion is very common.
3. The joints affected are not necessarily symmetrical.
4. Pain is usually severe, and is not relieved by the Aspirin group of analgesics.
5. Once the joint has been affected, there is usually some permanent damage: in acute Rheumatic Fever on the other hand, the joints are rarely permanently affected, and there is greater evanescence.
6. The patient has a toxic look and is usually running a temperature.

The individual features are as follows:-

(a) Non-Specific

i. Foci of infection are regularly demonstrated in patients with this type of arthritis.

ii. Signs of sepsis may be present in the teeth, nose and throat, (tonsils, paranasal sinuses); bowel, (appendix, gall-bladder); and genito-urinary system (prostate, vesicles, cervix).

(b) Specific

i. The joint changes are usually part of a general toxaemia, and are not the primary manifestation of the disease.

ii. Any joint may be involved, but it is usual to have certain parts picked out, more often in certain infections, e.g. in gonorrhoea the temporo-maxillary joint, and the sterno-clavicular are always mentioned.

From my own experience in Gonorrhoea, the knees and ankles have been by far the most frequently affected.

A list of the specific diseases which produce joint lesions under this heading follows:-

Tuberculosis.

Streptococcal infections (scarlet fever).

Pneumococcal infections.

Dysentry.

Gonorrhoea.

Syphilis.

Leprosy.

Typhoid Fever and Paratyphoid Fever.

Malta Fever (Brucella Melitensis).

The former group (a) is the infective arthritis of the undifferentiated type, or focal arthritis, while the latter (b), includes the frankly suppurative arthritis of the pyogenic streptococci and the staphylococci.

2. TRAUMATIC ARTHRITIS.

The effect of trauma is well known as a causative factor in the production of an arthritic

joint. In an analysis made by Dr. Figdor ③ of some 480 cases, something like 267 gave a history of previous injury of the joint. Any undue stress or strain in a joint may determine the onset of rheumatic changes. These changes are, of course, much more liable to occur in anyone who possesses the rheumatic diathesis. An example of this is well seen in the knee joints of patients who suffer from flat foot. In drop foot it is obvious that the knee joint is functioning under abnormal mechanical stress.

3. DEGENERATIVE ARTHRITIS.

This is more of a joint disease than a general disease (c.f. Rheumatoid Arthritis).

In this group the first obvious sign is that there is no inflammation. The disease has had a slow and gradual onset. One joint is usually picked out and that a major joint. The first two clinical features are stiffness and pain. It is in this group that Heberden's Nodes are found and also the osteophyte.

Another interesting feature of this senile type is that true bony ankylosis never occurs. Any locking of a joint that takes place is due to loose fringes of bone or cartilage or marginal osteophytes.

The patients in this group are of the thick-set

type, inclined to be overweight, have a poor circulation, and very often an increased blood pressure. In addition, there is usually some endocrine disturbance. This disturbance is a hypothyroid one,(4) and where this occurs the administration of thyroid extract usually does a great deal of good. Cases of Arthritis at the menopause come under this heading, and in them the endocrine disturbance is usually a mixed one. The thyroid, pituitary and ovarian glands are not functioning in harmony. At this time of life there is usually an increase of weight and this acts in a traumatic sense in so far as the knees are concerned.

The Villous type of Arthritis which is classified in the British Medical Association Report, comes under this heading. McCarrison observes that in the goiterous districts of the Himalayas a co-existence of Arthritis and Goitre is so common as to suggest a similarity of origin. The knees are the site of predilection, and in the early or villous stage, osteophytes are absent, while in the advanced stages the changes typical of Osteo-Arthritis supervene.

Hip. In the case of Osteo-Arthritis of the hip, the aetiological factors concerned in the production of the various types described are traumatic, mechanical, toxic, chemical, local disease of the articular ends

of the bone.(5) Once degenerative changes set in, alterations occur in the shape of the component parts of the joint, so that whatever the original cause, there is an additional mechanical factor involved.

4. METABOLIC ARTHRITIS.

This group includes:

(a) Gout, and
(b) True Rheumatoid Arthritis.

(a) Gout.

This is more common than is usually recognised. Out of 5,000 cases attending the British Red Cross Clinic, 2.9% proved to be of this nature. An inherited error in metabolism is usually found; also it is very common to find a hereditary history of gout in the family. The blood uric acid is usually above 3 mgs.% and during an acute attack, it can rise as high as 9 mgs.%. There appears to be a breakdown of one's own tissue proteins and these act as antigens. Thus a condition of auto-sensitisation occurs. The typical swellings (tophi) which occur in and around the joints are composed of bi-urate of soda, uric acid crystals, calcium urate and occasionally calcium phosphate. These deposits also occur in the phalanges and appear radiographically as the typical punched-out areas. These will be discussed in detail when the X-ray appearances are considered.

One of the interesting points that have been discovered by the routine examination of the standard hand, is the frequent occurence of a "gouty diathesis" in cases of so-called chronic arthritis. In my own series of cases two such examples are given. These patients had been, for many years, to various spas and had courses of baths and various treatments, including vaccines. While they derived temporary benefit, I think that specific diet and drugs for the underlying gouty condition would have been of great value. In neither case had any classical acute attack of gout been experienced.

An important clinical observation (6) is the frequent association of degenerative and hypertrophic signs in almost every one of the cases of Gout analysed by Strangeways.

It is still contended by many, that true gout is a purely metabolic disorder and that it has no connection with Arthritis. This view I think is correct were only the acute classical type considered, but where you get recurrent attacks, the irritation set up by the gouty deposits in and around the joint, sets up an arthritic process. In all the chronic cases of gout I have observed, there have been arthritic changes around the joint, seen radiographically. This supports the pathological findings of

Strangeways. Furthermore, in chronic gout, complete ankylosis of a joint does occur as a terminal sequel.

An interesting speculation and probable deduction is that in those cases of chronic degenerative arthritis where there is cartilagenous destruction and ankylosis of the joints, there is a metabolic disturbance - a disturbance of the purin metabolism, in other words, gout.

The opposite conception,(7.) that the urate deposits are a result of a rheumatic joint inflammation, is not supported by clinical observation.

These exponents consider that abnormal food proteins exist and act in an allergic manner, in a similar way to bacterial products.

(b) Rheumatoid Arthritis.

The clinical picture of this group is very typical. It occurs practically only in females, and the age incidence is the reproductive period. Women who are predisposed to Rheumatoid Arthritis are usually thin, pale and of the aesthetic type; they have poor digestive organs, suffer from lassitude and are of a worrying nature. The initial attack is usually ushered in by slight stiffness and fusiform swelling of the phalangeal joints. A notable feature is the symmetry of these swellings.

Very often there will be a slight rise of temperature. On a thorough examination of these patients, no signs of infection can be detected. These patients sweat easily, and there is a generalised wasting of the muscles and skin. It is from these features that the word atrophic is used. The blood pressure is low and the pulse is increased. These patients have a poor sugar tolerance and hence the usefulness of insulin in the treatment. At certain stages of the disease, an interesting biochemical fact is that, if a sample of venous and arterial blood is taken, the same reading of blood sugar is registered.(8) This would indicate that the tissues are unable to utilise sugar. A low carbohydrate diet is, therefore, most useful in the treatment. The capillary circulation is poor, giving an appearance similar to Raynaud's syndrone. There is a vaso-constriction due to disturbance of the sympathetic system. This action is a result of endocrine imbalance or the effect of the abnormal calcium metabolism influencing the Vegetative Nervous System. This clinical picture is similar to a case of Grave's disease.

It is in Rheumatoid Arthritis, that the Blood Sedimentation Rate ("B.S.R.") gives the highest reading; in many case the reading shows the possible maximum in thirty minutes. This test, along with the standard radiograph hand, gives the most useful information about the progress of a case.

The modern conception of Rheumatoid Arthritis is that we are dealing with a metabolic disorder. The sweating

and tachycardia are due to a raised basal metabolic rate.

In a series of cases which were investigated this was found to be from 15 to 20% above the normal. To compensate for this raised metabolism, an adequate diet is essential in treatment, and also the importance of looking upon this particular form of arthritis as a metabolic disease should be kept in mind.

Treatment should be directed against the general condition of the patient and not merely concentrated upon the local joints involved.

An arthritic process is brought about by infection, trauma, metabolic disorder, degenerative changes, and, once established, the boney changes are dependent upon the activity of the various factors discussed in the preceeding pages.

These factors are so variable and do not lead to any degree of exactitude that a clinical or aetiological classification of the chronic arthritic joint from this aspect is too indefinite.

But, in radiology, we have a means of determining the pathological process which is predominant and hence an exact basis for classification is present.

PHYSIOLOGY AND CHEMISTRY OF JOINT STRUCTURE.

The component parts of the moveable joint, (diathrosis) are, the articular capsule, the synovial fluid, the cartilage and the bone.

The articular capsule is composed of two parts:- the fibrous stratum and the synovial stratum, or more commonly, the synovial membrane. The fibrous stratum is the essential bond of union between the bones.

ARTICULAR CAPSULE

Synovial membrane is derived from the mesenchyme. It has the power of regeneration and secretes fluid mainly derived from the blood plasma, modified by mucin. Certain salts may be found in the synovial fluid which are, for the most part, phagocytes and help in removing particles from the joint. There are also lymphatics which aid in draining the joints. Diffusible substances pass easily from the blood to the synovial fluid.(9.) It has been found that, following glucose injection, the sugar concentration of the synovial fluid promptly rises to a level as high as, or higher than, the venous blood. This assists the nourishment of the cartilage which has a content of glycogen.

CARTILAGE.

This is only modified connective tissue. There

are two varieties: hyaline and white and yellow elastic fibro-cartilage. Hyaline articular cartilage has three separate zones:- a central zone, a peripheral zone and a cartilage-bone-portion. The peripheral portions are covered with a vascular membrane. At the margins of the cartilage is the "Circulus articuli vasculosus" of Hunter. The peripheral portions are nourished from this zone. There are no blood vessels or nerves in cartilage.

The Calcification of Cartilage.

Histological changes. (10)

In all cases of calcification of cartilage the following sequence of histological changes appear:- This is the normal process of development and also occurs pathologically.

i. The calcium salts always appear first in the form of granules.

ii. These granules appear first around the cartilage arc and are imbedded in the matrix immediately outside the cell capsule.

iii. The salts appear before blood vessels can prevent calcified areas.

iv. The calcium salts do not alter after changes have occurred both in the cartilage cells and in the matrix of the area to be calcified.

BONE.

That portion of the bone which is subjacent to the cartilage is called the subchondral plate (11) and is cancellous in character.

The changes in bone which accompany disuse are those of atrophy. This is evidenced by fewer and thinner trabeculae and thinning of the cortex.

The synovial membrane and bone marrow are the tissues which primarily respond to the activities of bacteria, toxins and traumatism. In all cases of Arthritis, except the degenerative type, the disease process primarily affects the synovial membrane and the marrow of the bone. Each of these tissues undergo proliferative changes. All other changes, atrophy, hypertrophy, loose body formation, eburnation and cartilage destruction, are secondary changes.

THE PATHOLOGY OF ARTHRITIS.

When we come to consider arthritis from the purely pathological standpoint, the subject divides itself into two main groups. These types are known by different names in different countries and it is due to this, that a great deal of the confusion in nomenclature has arisen. They are as follows:-

1. Osteo-arthritis - hyperthrophic or degenerative.
2. Rheumatoid Arthritis - atrophic or proliferative.

I propose now to discuss the pathology of each of these two groups, bearing in mind that the aim of radiological diagnosis is the exact interpretation of these

pathological changes, some of which it is impossible to detect radiologically, especially in the earliest evidence of disease, which occurs in and around the joint.

OSTEO-ARTHRITIS.

Dr. Thompson,(12) in his work on Arthritis, is of the opinion that the primary process is waste of bone tissue, followed by a secondary attempt to repair the destruction by throwing out of new bone of irregular structure. Such degeneration may be due either to an inadequate blood or lymph supply.

Timbrell Fisher(13) has pointed out that Osteo Arthritic changes tend to originate at the central part of the cartilage, which is the leased nourished.

The degeneration of cartilage in osteo-arthritis can be brought about by various agencies - trauma, pressure of bony tumours, gout, diseases of the central nervous system and causes suggesting deranged metabolism. Whatever the original cause, the arthritic process tends to continue.

Prolonged unresolved inflammation of all joint surfaces are bound to interfere with the proper vascular and lymphatic circulation in the joint.

The first change in this type occurs in the cartilage and this is a primary degeneration. This occurs in the centre of the cartilage where maximum

pressure and friction take place. The cartilage becomes softened and eroded. This breaking down is patchy in nature. As the disease progresses, you get splitting of the cartilage and exposure of the bone surface. These denuded areas of bone react by becoming thickened or sclerosed; likewise a compensatory overgrowth of cartilage occurs. Thus the line of articulation becomes very irregular. These bony and cartilagenous outgrowths at the margin of the articular cartilage, produce the spurring, lipping and exostosis. This lipping may consist of new cartilage formation as the lateral portions of the articular cartilage are very vascular: or, it may result from the development of cartilage in a fold of synovial membrane and this becomes ossified.

Heberden's Nodes (14.), on the other hand, originate from the periosteum and are due to it's proliferation.

The cancellous bone at the articular end, called the subchondral plate, becomes greatly thickened and the marrow spaces nearly obliterated.

From constant friction this part of the bone becomes highly polished and it is then said to be eburnated (X-ray shows as a thin white pencil line). This eburnation only occurs in Osteo Arthritis and never in Rheumatoid Arthritis. True bony ankylosis does not occur in Osteo Arthritis. The description

of the gross histology by Nichols and Richardson,(15) is well worth quoting: "The joint cartilage is irregular in thickness and markedly so in structure. It is fibrillated and calcified. Its cells are swollen. Parts of the cartilage may be ossified. The underlying bone is thickened and dense. The marrow is fatty and fibrous. The synovial membrane is thickened and fibrous and is thrown into villi. Several observers have noted cyst formation in the bone and in the region of the joint".

Special mention must be made of these cysts which occur chiefly in the head of the femur. Dr. Alec Thompson(18) maintains that these cyst-like structures are present in all cases of osteo-arthritis of the hip and are pathognomonic of this disease.

Dr. Gilbert Scott, on the other hand, considers that these cavities in the head and neck of the femur may be of gouty origin.

RHEUMATOID ARTHRITIS.

The first changes to take place are swelling and oedema of the soft tissues around the joint. The capsule of the joint becomes engorged and vascularised. Round cell infiltration occurs in the synovial membrane; this takes the form of focal collection of lymphocytes. This process tends to

proceed rapidly and causes a layer of granulation tissue to be laid down and this spreads inwards over the articular cartilage in the form of a pannus.(11) New destruction of the cartilage is brought about by the pannus and also subcartilagenous granulations are formed. Predominance of this change leads to fibrous ankylosis.

Proliferative changes are also occurring in the subchondral plate. Vascularisation of the connective tissue elements in this part of the marrow takes place and this process extends upwards into the joint. This connective tissue contains osteoblasts and eventually, by the destruction of cartilage, fuses with the pannus above.

Thus this osteoid tissue proliferation may give rise to the formation of true new bone with subsequent bony ankylosis. Cartilagenous ankylosis is brought about by proliferation of the perichondrium. All these pathological changes may be going on at the same time in the joint and it is the predominance of the one or the other which will determine the final result. Strangeways(16) describes six varieties of this Rheumatoid group. They are as follows:-

1. Capsular Type. The changes (thickening and fibrosis) are practically confined to the capsule of the affected joint.

2. Dry Type. The characteristic feature is the absorbtion of the synovial fluid, the

atrophic, fibrous and tightly contracted capsule gripping the bone firmly and causing the articular surfaces to lie in close contact.

3. The Adhesive Type. This begins with inflammatory changes in the synovial membrane and capsule, going on to the replacement of cartilage by inflammatory connective tissue which becomes fibrous and ends in bony ankylosis.

4. The Atrophic Type. This is marked by great rarefaction of bone and much fatty change in bones, muscles and other tissues. The capsule, synovial membrane and articular cartilage atrophy and as the early inflammatory changes subside, bony ankylosis often occurs.

5. The Villous Type. Swelling is chiefly due to numerous pedunculated villi, though increased fluid may be present. Melon seed bodies are found.

6. The Infective Type. These cases clinically resemble the adhesive type and are usually due to some micro organism, e.g. gonococcus, pneumococcus, dysentry bacillus and streptococcus and staphylococcus.

Occasionally suppuration develops and pus appears in the joint. It has lately been discovered that the synovial fluid has chrondolytic properties which tend to prevent fixation. The synovial fluid in this type contains many polymorphonuclear leucocytes.

It is in the Rheumatoid Group that important alteration in the bone calcium takes place; in the long bones decalcification and absorption of the osseous framework occurs. The lacunar spaces are wider than normal. This question will be discussed further under the radiological changes. Also the muscles become wasted. This wasting is not entirely

a disuse atrophy. The muscle fibres become invaded by round cell infiltration; patchy fibrosis develops which gives rise to adhesions and thus the typical flexion deformities are produced.

It will be noticed that the Villous type is regarded by Strangeways as a variety of the Rheumatoid group and not of the degenerative group.

THE TRAUMATIC TYPE.

An important clinical group, the Traumatic, requires special mention before I leave the subject of pathology.

Whenever a joint is submitted to trauma, damage may take place in any of the compnnent parts of that joint. The histopathology is as follows:-

The Synovial Membrane. Acute trauma will produce haemorrhage, with perhaps rupture of the synovial membrane and capsule, followed later by oedema. Fibrosis may occur but never lymphocytes in the focal collection as seen in Rheumatoid Arthritis.

Cartilage. Here there is surface fibrosis, myxomatous degeneration of the matrix, replacement fibrosis and calcification.

Bone. Increased calcification of the subchondral plate and varying degrees of fibrosis of the contents of marrow spaces in the cancellous bone occur.

THE RADIOLOGICAL ASPECT OF RHEUMATIC ARTHRITIS.

By this means you can obtain direct evidence of the underlying pathological changes. The essential feature of this examination is the interpretation of pathological changes by means of shadows.

The use of X-ray examination is helpful in five different ways:-

1. The differential diagnosis from other diseases involving bones and joints.
2. To distinguish the varieties of Arthritis.
3. To determine prognosis.
4. To determine the activity of the disease.
5. To determine the state of the joint.

The use of X-ray examination, particularly of the hand, has also been of value in correcting diagnoses of clinical arthritis; on radiological examination the correct interpretation has been discovered.

The following is a list of conditions where errors were made, in order of their frequency:-
These cases were clinically diagnosed as arthritis.

Secondary carcinoma of the spine and pelvis.
Paget's disease.
Charcot's Joint.
Carcinoma, (femur and humerus).
Myelatemosis (multiple tumour of the red bone marrow, mostly affecting spine and ribs), and enchondromata.
Renal Tuberculosis and Calculus.
Dislocated humerus.
Needle in knee joint.

THE SIGNIFICANCE OF THE STANDARD HAND. (17 & 18)

By the term standard hand, as applied to the X-ray film, is meant that all the technical radiological factors are constant. In my own series of cases, I have used a tube distance of 18" with 60 K.V. and 10 M.A.; the tube has been centred over the middle metacarpo-phalangeal joint; the exposure time has been one second and the films have been developed at 65° Fahrenheit for five minutes duration; no intensifying screen was used.

The hand was chosen for the following reasons:-

1. It is easily accessible and therefore a quick examination can be done and only a small film is necessary; this will naturally reduce the cost.

2. A clear picture can always be obtained as the bones and joints are in such intimate contact with the films and there is a fairly constant average thickness of tissue.

3. Little soft tissue intervenes between the film and the bone, and where density of the bone has to be gauged, this is of paramount importance.

4. The hand is rarely the site of disuse atrophy.

5. Radiological examination of the hand often gives the key to the diagnosis in any major joint that may be involved.

I will now discuss the various forms of Arthritis in order, and describe the radiological changes which occur in each separate group. Before doing so, however, there are three radiological terms which require some explanation; these are Osteoporosis,

Osteosclerosis and Bone Density Balance.

OSTEOPOROSIS: is an increased transparency of the bone or diminished bone density; in other words, there is decalcification of the bone. It can be:-

a) generalised.
b) localised.

The generalised osteoporosis is seen in hyperthyroidism, hyperparathyroidism and rheumatoid arthritis and indicates a disturbance of calcium metabolism.

The localised is seen in active infective conditions such as tuberculous caries, ostomyeletis and leprosy. As healing takes place a local osteosclerosis results.

OSTEOSCLEROSIS: is an increase of the normal bone density; it is an inflammatory reaction and is Nature's method of repair. There is an increase of bone calcium.

BONE DENSITY BALANCE: This is a term which is used to denote the relative density of the various parts of a bone and is best seen in long bones. The density of the cortex is normally three times that of the medulla. This relationship should remain constant even in extreme degrees of osteoporosis or osteosclerosis. In Paget's disease where there is an increased density of the bone, the Bone Density Balance is upset. The bone, in extreme degrees of this complaint,

is of a uniform density throughout. The antithesis of this can also occur. There may be an increased transparency but still the cortex should be as three to one provided no abnormal metabolic process exists. It would appear that the Bone Density Balance is only altered when a generalised calcium upset is present. Its exact significance is, however, "sub judice".

RHEUMATOID ARTHRITIS.

As this disease invariably arises in the phalanygeal joints, most of my cases are limited to the X-ray appearances of the hand.

The radiological appearances can really be grouped under three headings according to the stage of the disease.

(a) In the very early stages where only inflammatory hyperplasia in the synovial membrane has occurred, there may be no change visible. Where slight effusion has developed in addition, there will be haziness of the joint outline. The first change seen in the bone is atrophy. This atrophy is the fundamental change and is a loss of bone calcium as evidenced in the X-ray by diminished bone density. This occurs in all the bones of the skeleton if the disease is active and progressive, but is best observed

in the hand. In addition to the general osteoporosis, there is also a local irregular distribution of the calcium in the bone. In the very early cases this is the only change that occurs.

The generalised osteoporosis is an indication of activity and illustrates the disturbance of the calcium metabolism that exists in this disease.

The Bone Density Balance is also altered. The significance of this is obscure but it is this feature that strongly suggests that Rheumatoid Arthritis is primarily due to a disturbance in Metabolism. As the disease improves, so also does the Bone Density Balance and this is used as an index of progress.

It would appear that there is a definite circulation of soluble bone calcium. The trabeculae of the bone remain unchanged, but the calcium and phosphorus salts ebb and flow.

b)

The second stage that occurs is a loss of cartilage. This erosion of cartilage is shown on the X-ray picture by a narrowing of the joint space.

As the disease advances, erosion of the articular end of the bone takes place. The

subchondral plate undergoes rapid decalcification. This is a similar change to that which takes place in chronic gout, but in gout you never have any general alteration in the bone calcium.

(19) The new bone formation which fills up the space left by the destruction of cartilage is of poor quality and appears as a coarse wide network.

(c) The last stage of the disease occurs where you get subluxation of the joints and fusion of the carpal bones.

True bony ankylosis thus occurs in this picture.

The more transparent the bone, the more active the disease. When the radiograph of the hand shows a return in bone calcium, you know that the case is improving.

G O U T .

In the early acute stage of gout no radiological change is seen. During the acute attack of gout, salts of uric acid are deposited in the articular ends of the bones and the surrounding tissues. This deposition, in the case of cartilage, being greatest at the surface, and usually absent at the central zone.(20)

In the chronic articular cases of gout, on the other hand, radiography is capable of demonstrating an exact and certain diagnosis.

The salts cast a shadow on the radiogram, the density of which varies directly according to the amount of calcium present. The salts of calcium and magnesium are denser than the other urates. These deposits produce clear-cut circular areas in the bone, which are very typical; they vary in size from minute pin-head spots to larger areas which may occupy the diameter of the bone.

It is an interesting consideration as to whether the typical punched-out areas can arise without any clinical sign or symptom in the hand. If they can, then we have a quick and easy method of determining those patients with a "gouty" diathesis.

These rarified areas in the bone are replaced by biurate of soda, and later on by granulation tissue. The punched-out-areas are very minute in the early stage. The hand, and particularly the bones of the metacarpals, is the commonest part to show these punched-out areas and they are only rarely seen in a major joint.

These circular areas are seen most commonly at the base of the terminal phalynx and head of the middle phalynx and lateral border of it's base.(21) The

relative position of these areas in gout is an important point in the differential diagnosis. There are many other diseases associated with translucent areas in the phalanges. I will now append a list of these and add a few notes where there is anything of interest or import.

1. Chondroma, enchondroma, myochondroma.
2. Implantation Dermoid.
3. Carcinoma deposits and Grant Cell Tumours.
4. Raynaud's disease.
5. Sclerodermia.
6. Fibro-cystic disease.
7. Traumatic - in a radiological examination of the hands of workmen who had been employed on the pneumatic road drill, all showed clear circumscribed areas in the phalanges and carpals.(21) These are apparently brought about by small haemorrhages and lend support to the theory that trauma could produce a similar condition in the hip joint.
8. Leprous osteitis multiplex cystica - large nutrient foramina are frequently present. A further description of this disease appears later on in the Thesis.
9. Lupus Pernio (osteitis Tuberculosa multiplex cystica). In this condition a trophic disturbance is responsible for the cystic areas; a similar state also occurs in cases of Leprosy.
10. Boecks Sarcoid.

This is rather a formidable list, but from a practical point of view, these are diseases associated with radiological changes and their clinical diagnosis has already been established.

The finger joints give the best indication of Gout in an individual. Here the earliest evidence of uratic deposit will be found on the sides of the phalanges. In addition to this, the articular cartilage becomes more opaque to X-rays.

Another feature, and one which is probably more common than the actual vacuole, is the notch, or what the French call the "Encoche en hallebarde".(22) This is a nick, present at the lateral extremities of the articulation; its outline is very sharp on account of the sclerosed bone which surrounds it.

The terminal phalynx very often has a very marked dentate appearance or crenated outline.

Further changes that take place are mottling of the carpals, loss of cartilage, particularly where tophaceous deposits have occurred near the joint.

Later on you get complete destruction of the articular cartilage. This is similar to that which occurs in Rheumatoid Arthritis but there is no change in the bone calcium.

Eventually subluxation occurs and fibrous ankylosis takes place, particularly in the little finger.

There are two main types from the radiological point of view:-

(a) Where you get enormous deposits of tophi but little change in the joint.

(b) Where few clinical signs are present but there is marked destruction of articular bone.

Thus it may be that the key to the diagnosis of a major joint may be revealed by the X-ray examination of the hand.

Two very interesting cases are cited in La Presse Medicale,(22) which lend support to the main theme of my Thesis.

The first of these concerns a man aged 54, who had suffered from gout from the age of 39. He had very severe attacks of gout in his big toe, laying him up at times for as long as seven months; he developed a Hullux Valgus deformity, and a surgeon decided to operate on his toe, without however, having the joint radiographed. When the incision penetrated the peri-articular tissues 15-20 c.c. of a whitish semi-purulent material oozed out. This proved to be a large soft Tophus.

The other case is that of an alcoholic subject who had suffered from a severe neuritis of the ulnar nerve and which had resisted all treatments for five months. A radiograph of the hand showed, at the tips of the fingers, changes characteristic of gout. Suitable treatment was given and a rapid improvement of the neuritis resulted.

There is a condition which has recently been reported under the term "Hypodermolithiasis",(23) and this has a clinical resemblance to Gout.

The resemblance is only clinical, as an X-ray examination will immediately establish the diagnosis.

Superficial calcareous concretions are found in the fingers, and practically all the cases that have been reported have occurred in patients suffering from chilblains, or with marked Raynaud's disease. In one case the fingers had the appearance of sclerodactylia.

The actual concretion mainly consisted of calcium phosphate.

The X-ray examination of the hand is reported to show a normal bone density; the calcium and phosphorus content of the blood was normal in one case reported by Ross.

In the three cases illustrated in this article,[23] I think there is definite evidence that the bone calcium is not normal.

Case (3) shows general osteoporosis, while case (2) has the appearance of increased bone density particularly of the middle fingers.

The appearance may be deceptive owing to different technique in each film, but it only illustrates the importance of a standard technique when it is necessary for comparisons to be made, and more so, where bone calcium estimation is under consideration.

This condition is of interest because of the

clinical resemblance to gout, and also to support my contention that a standard radiogram of the hand is a valuable aid in diagnosis.

OSTEO-ARTHRITIS.

The characteristic change here is the production of new bone - what is commonly called the "plus bone" arthritis in contra-distinction to the Rheumatoid group, which is called the "minus bone" arthritis.

This group in the past has been used to cover a very miscellaneous collection of chronic articular disease. As the earliest change in Osteo-arthritis is an erosion of the central part of the articular cartilage, this is not readily seen radiographically. The first change seen then by means of X-ray examination is the periarticular lipping. This lipping is produced by outgrowth of ossified cartilage.

Here the radiogram is sharp and clear, as there is no fluid to obscure, and the increase of bone calcium which is usually found, adds to the sharp outline. This is in marked contrast to the picture in Rheumatoid Arthritis.

Loose bodies in the joint are seen in Osteo-Arthritis and these result from detached particles of cartilage or bone or both, becoming free in the

joint. They are usually derived from periarticular chondro-osteophytes. They are the so-called "joint-mice".

Osteo Arthritis is essentially a degenerative condition. The primary process is a waste of the bone tissue followed by a secondary attempt to repair the destruction by throwing out of new bone of irregular structure. The new bone gives rise to the production of osteophytes.

The limitation of movement in this group is due to these osteophytes.

The radiological changes of the hand, the hip and the spine are worthy of special notice.

THE HAND.

Here we first of all get loss of cartilage and this usually occurs at the tips of the fingers. Pain in the joint is the first sign in this disease and is a result of this destruction. The joints first involved are dependent upon the work which the individual has been in the habit of doing. For instance, in the tailor the index finger is usually first affected, whereas in the seamstress it is usually the first joint of the middle finger. These changes are brought about as a result of constant strain. Narrowing of the joint space

is followed by erosion of the articular bone with consequent deformity.

This is usually a lateral displacement. New bone is laid down and this is clinically seen as Heberden's Nodes. They are commonly seen at the base of the terminal phalynx. The appearance is as if the bone had been "splayed-out".

There is one other characteristic feature which has been described by Dr. Gilbert Scott as the peri-articular ossicle. The ossicles usually occur around the capsule or ligament of the joint and are commonly seen at the terminal joints of the first and second fingers. New bone appears in the soft tissues independent of the parent bone.

In osteo-arthritis of the fingers the ends of the bones are dentate but movement is not much interfered with. Radiologically the condition looks very advanced and appears as if no movement were at all possible in the joint.

It is in this particular type of arthritis that the use of the X-ray of the hand is so valuable. Suppose a major joint is the seat of arthritis and there is no evidence in the hand of osteo-phytes, then it is probable that the arthritis

is an infective type or is not a true degenerative osteo-arthritis.

These particular changes have the appearance of chronic gout.

THE HIP:

There are many variations of osteoarthritis in the hip joint. In many cases no osteophytes can be seen. The head of the femur appears to bore its way through into the pelvis.

The first change is a loss of cartilage at the maximum pressure points.(5) This shows itself on the radiogram as a loss of joint space which may be localized or may be evenly distributed.

Following this, there is displacement of the head upwards and sometimes at the outside of the acetabulum. When this occurs it gives the appearance of dislocation. The acetabulum then becomes filled with new bone.

The third change is the fringe. This fringe is a result of the deposition of calcium in the periphery of the acetabulum and typical osteophytes are produced. These osteophytes interfere with movements of the joints.

An example in this group is what is sometimes called "Grinder hip". Here the head of the femur grinds its way into the acetabulum en masse and gives the appearance of fusion.

The later changes which occur in Arthritis of the hip joint are alteration in the shape of the neck; the neck takes on a mushroom appearance. Following this, sclerosis of the bone occurs around where the cartilage is devitalised. This is in a sense a defensive mechanism. At the same time we see osteo-porosis of the head and acetabulum, which is usually irregular and patchy.

There are two opposite changes taking place in the joint simultaneously. It is probable that this state of affairs gives the appearance of multiple holes around the femur and pelvis. The changes in the hip are more suggestive of localised blood borne infection; one particular bone or joint is involved, as in osteomyelitis. There is a localised osteoporosis which later may give rise to a sclerosis. This sclerosis is of a reactionary nature.

Dr. Alec Thompson (12) discusses the radiological sides of Chronic Arthritis. He maintains that cyst-like structures are seen in all radiograms of this disease (osteo-Arthritis of the Hip).

It is impossible to tell from radiograms whether these cysts contain granulomatous material. They vary in size and the appearance of these cysts is de-

termined by the surrounding Osteosclerosis.

There are apparently four distinct types of Osteo-Arthritis of the hip, depending on the origin of the disease:-

1. In the majority of the cases, the primary focus of the disease is in the Ileum at the upper and outer edge of the acetabulum. This type produces shortening of the limb and eversion.
2. Where the primary focus is in the head of the femur.
3. Where the head of the femur appears to enter the acetabulum up to the limit of the trochanter.
4. Where a great deal of moulding takes place between the acetabulum and the head of the femur. Here the cyst-like forms are widely distributed around the whole joint. When acute clinical symptoms subside it will be found that sclerosis is taking place around the joint surfaces.

Thompson maintains, that osteophytes are not an essential feature of this disease, but looks upon osteophytes as a reaction of nature to limit the progress of the disease.

It is possible that some cases of Osteo-Arthritis of the hip are really due to gout. Dr. G. Scott has suggested that these cysts are really in some cases tophi and filled with biurate of soda. The use of

the X-ray of the hand would here be of value, as it would be a strong point in favour of gout were the typical appearances of gout present in the hand.

THE SPINE:

Spondylitis.

There are two main types of arthritis in the spine and each type is known by various names. They are as follows:-

i. Bechterew's arthritis.
Spondylitis adolescens.
Spondylitis ankylopoietica.
Acute spondylitis.
Marie-Strümpell Arthritis.
Poker Spine.
Bamboo spine.

ii. Spondylitis osteo-arthritica.
Spondylosis.

They are two distinct diseases and correspond to the acute Rheumatoid type and the chronic hypertrophic type respectively. The acute spondylitis usually occurs in young people and according to the researches of Gilbert Scott, it is usually preceded by inflammatory changes in the sacrum and sacro-iliac joints. The chief radiological feature is the early ossification of the ligaments and discs. There is an osteoporosis of the vertebral bodies and thus softened they are compressed one into the other. The ossified ligaments appear as straight lines and the spine becomes a rigid stick, curved where

greatest pressure has taken place in the vertebrae. This gives rise to the so-called "tram-line" appearance in a skiagram. No spur formation takes place in this type.

Spondylosis, on the other hand, is a degenerative condition and osteophytic formation is common. These hypertrophic fringes on the margins of the bones can produce fixation of the spine but in a different manner to the fixation in acute spondylitis. These small outgrowths from the edges of the Vertebrae have been likened to parrots' beaks. Evidence of hypertrophic arthritis will usually be found in the hand as well, and there is often an arthritis of the sacro-iliac joint.

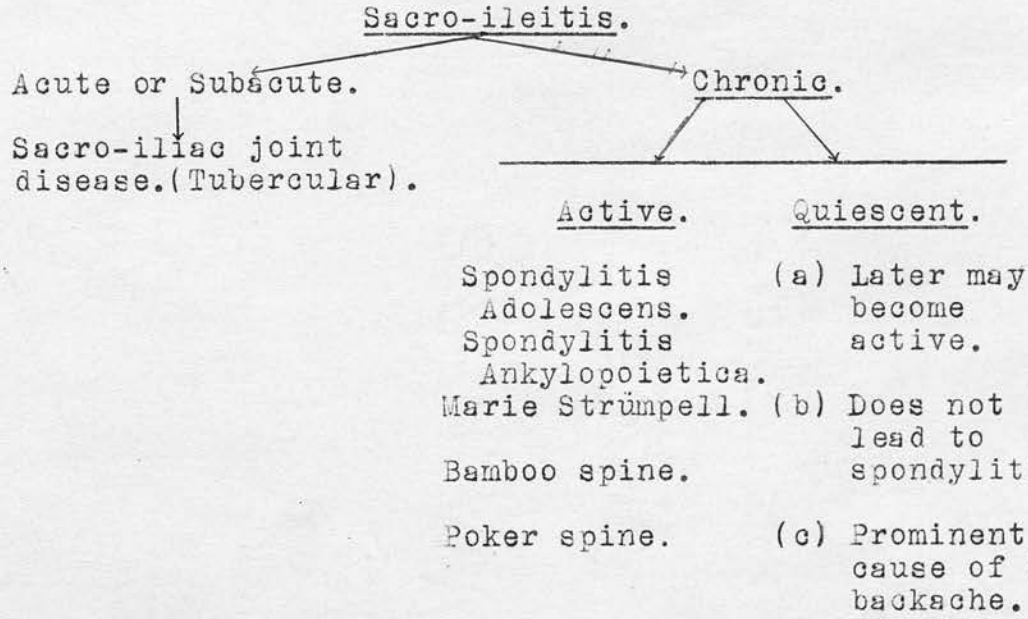
An interesting piece of research work that is at present being carried out at the Red Cross Clinic, is well worth mentioning.

In tracing back the earliest evidence of disease in these acute adolescent cases of Spondylitis, it has been found out that they all have the initial disease process in the sacro-iliac joint. The early clinical history is usually the same; they give a history of growing pains or muscular rheumatism about the shoulders and down the thorax, when about 16 or 18. This usually clears up, and

in a few months there is a return of the fibrositis to the knees, thighs and then to the back.

The investigation suggests that a chronic infection of the sacro-iliac joint is a precursor, and a cause, of the acute spondylitis. One hundred and ten cases have been collected and an examination of the films readily shows disease of the sacro-iliac joint. The difficulty, of course, has been in getting a radiograph of the sacro-iliac joint at an early stage. Pain over the joint is not a symptom as it is in tubercular disease, and it has only been discovered by the routine use of the X-ray in cases giving a suggestive history.

The following represents diagrammatically the course of the disease process.



The differential diagnosis between the two types is as follows:-

In tuberculous disease the affection is unilateral, whereas in the spondylitis type, it is bilateral.

Pain is a constant symptom in tuberculosis, but in the active type of sacro-ilietis there is no pain over the joints; it is usually of a referred nature. The tubercular disease goes on to abscess formation, but the other goes on to bony ankylosis.

The sacro-iliac joint is composed of an oval cartilagenous part, and a larger fibrous part. The cartilagenous part is first affected in Tuberculosis and the disease spreads upwards; in infective sacro-ilietis the disease process starts in both tissues simultaneously.

The presence of a low blood phosphatase in spondylitis ankylopoietica is difficult to explain in view of the excessive calcification in the ligaments, and the deposit of new bone.

The constant association of this sacro-iliac disease with acute spondylitis is a very interesting consideration.

In view of the hopelessness of the prognosis in acute spondylitis; and the terrible crippling which it produces, one should keep the possibility of this

in mind when radiographic changes, such as opening up of the joint and fuzziness, are seen in the sacro-iliac joint in a young person.

INFECTIVE ARTHRITIS.

(Specific or non-specific.)

Usually one joint only is involved, and very frequently this is the wrist. Here again the bone calcium is normal and only that part which is affected shows any radiological change.

There are no specific distinctive X-ray appearances.

The acute form will show soft tissue swelling and effusion in the synovial cavity. The joint space will thus be increased by separation of the articular surfaces. If this persists for several weeks, decalcification of the articular ends of the bones will occur, and there will be an early erosion of cartilage with narrowing of the joint spaces. The first change is a loss of cartilage without any other change in the joint. There will also be found multiple areas of rarefaction in the bone close to the affected joint.

If the disease becomes chronic a progressive atrophy of the bone will be seen, due to disuse; also there is a general destruction of the carti-

lage and bone, followed by a production of fibrous tissue or osteophytes. The new bone will be detected first of all at the attachments of the joint ligaments. The formation of fibrous tissue shows as a general clouding of the joint space.

The various types of infection produce similar changes to the above, and they cannot be distinguised radiologically, except perhaps, in tuberculosis.

The radiological appearance of the tuberculous joint shows more generalised atrophy. In tuberculosis all the bones in the neighbourhood of the diseased joint show decalcification. In Infective Arthritis, there is a local osteoporosis as opposed to a generalised osteoporosis which occurs in Rheumatoid Arthritis.

Most of the infective cases show definite osteosclerosis as well. This again should be looked upon in the nature of a reaction, and indicates that healing has taken place.

The appearance of osteophytes is also a useful diagnostic help in differentiating from Rheumatoid Arthritis.

BIOCHEMICAL OBSERVATIONS IN RELATION TO BONE PATHOLOGY.

CALCIUM METABOLISM.

It will be necessary to give a brief résumé of calcium metabolism to enable one to understand the changes which occur in the bones in Arthritis.

The most recent and comprehensive work on Calcium Metabolism has been done by Dr. Donald Hunter.(24) The Goulstonian lectures in 1930, given by Dr. Hunter, provide a standard work on calcium and, therefore, most of the information that follows is derived from this source.

NORMAL CALCIUM METABOLISM.

The richest source of calcium is the green parts of plants as opposed to roots. Man derives the greatest amount of his calcium intake from milk, cheese, lettuce, eggs, vegetables and nuts.

CALCIUM ABSORPTION.

The daily requirements of calcium to maintain health is one gramme per day when the sketeton is mature.

Calcium chloride raises the serum calcium more than calcium lactate. This is due to an acidosis which raises the serum calcium. The absorption of calcium is increased by any fat which contains

vitamin D. Any fat which contains ergo-sterol and has been activated by ultra-violet light, contains vitamin D.

EXCRETION.

Calcium is excreted by the intestine and by the kidney. On a normal diet the faecal calcium includes the unabsorbed and the re-excreted calcium. The urinary calcium varies from .1 of a gramme per day on a low calcium diet, to .5 of a gramme on a high calcium diet.

Acid diets increase the excretion of the urinary calcium.

An example of an acid diet is bread, meat, rice, fats, macaroni and sweets. The production of an artificial acidosis by means of ammonium chloride will give the greatest increase of calcium excretion.

CALCIUM CONDITION OF THE BLOOD.

Sixty per cent of calcium in the blood is in a diffusible form and 40% in a non-diffusible form.

The plasma and the serum have almost identical amounts of calcium. This varies from 9 to 11 mgs %. (The serum calcium is 1 mg. less in pregnant women). But of this amount probably only 2 mgs. exist as calcium ions in the serum, while 4 mgs. are in some form of combination with protein or possibly lipoids.

As long as this active fraction does not go below the normal 2 mgs.% no signs of calcium deficiency will be observed even though the total blood calcium is low.(25)

THE DISTRIBUTION OF CALCIUM IN THE TISSUES.

The calcium is distributed evenly throughout the soft tissues of the body, muscle tissue containing 6.5 mgs. per 100 grammes. The corresponding figure for the sketeton is 10,000 mgs; of the total calcium in the body, 99% is in the skeletal tissue including the teeth and 85% of this bony structure is a combination of calcium and phosphorus.

Calcium phosphate constitutes 80% of the mineral constituent of the bone. Calcium carbonate is present to the extent of 7.3 % and is responsible for the hardness of the bone.

According to Kramer, Calcium and Phosphorus in bone probably exist as dicalcium phosphate (Ca H Po4 2 H_2O).

THE FUNCTIONS OF CALCIUM.

1. Formation of bone. The skeleton acts as a reservoir of calcium and phosphorus. There is a continuous ebb and flow of calcium salts from the bone into the blood. The adult skeleton is not a fixed chemical formulae but is in a constant state of movement, particularly as regards the calcium content. An example of this - the hen can draw upon calcium from its bones to supply the shell of the egg if there is a deficiency of lime salts in its food.

2. The regulation in the blood and tissues of a balanced iron metabolism.

3. Calcium controls the contractability of plain and striped muscle.

4. Coagulation of the blood.

5. Transference of impulses at the neuro-muscular junctions and synapses.

SOURCE OF AVAILABLE CALCIUM IN THE BONES.

In starvation, sodium and potassium are retained, whereas calcium is excreted continuously from the bones. The spongiosa of the bone acts as the store-house of readily available calcium, while the corticalis is at first spared in the process of calcium metabolism. Experiments showed that in cats the trabeculae of spongy bone are diminished on a low calcium diet and increased on a high calcium diet. No gross change takes place in the cortical thickness. It is evident that there must be a very delicate mechanism which maintains a balance between absorption, deposition, and elimination, and there is a very intimate relation between the calcium and phosphorus of the blood.

NORMAL METABOLISM OF PHOSPHORUS.

ABSORPTION.

Phosphorus is ingested as nucleo protein (meat), phospho protein (milk), lecethin (egg yolk and liver), and as inorganic phosphorus.

PROPORTIONS IN THE BLOOD.

The plasma and corpuscles of the blood contain

equal amounts of organic and inorganic phosphorus. The inorganic phosphorus in the blood varies from 2.5 to 3.5 mgs.%. In children, where active ossification takes place, the blood phosphorus can rise to 5 mgs.

Only 1/12th of the total phosphorus in the blood is present as inorganic phosphorus.

The organic phosphorus is present as ester phosphorus and lipin phosphorus.

THE FUNCTIONS OF PHOSPHORUS.

Phosphorus controls the:

1. Utilization and storage of carbohydrates.
2. Phosphorus is essential to the deposition of bone.
3. Chemical changes which precede the contraction of muscles.
4. Acid-base equilibrium.

Its part in the production of ossification will require to be dealt with at greater length (see 2a).

(In carrying out these various processes, certain enzymes are essential. These enzymes are called Phosphatases.)

2a. OSSIFICATION.

The part which phosphorus plays in this process is closely linked to that of calcium. The actual deposition of bone is brought about by the precipitate

of insoluble calcium phosphate in the presence of phosphatase on a frame-work of osteoid tissue. There appears to be a reciprocal action between Calcium and Phosphorus which is endogenous. Thus, when there is a rise in the calcium ions of the blood, there is a fall of the Phosphorus and vice versa. The relationship holds good except where there is interference with the absorption of these elements, as in Rickets.

It is thus apparent that the metabolism of Phosphorus is of paramount importance in bone pathology.

PHOSPHATASES.

These are enzymes which hydrolise phosphoric ester, such as glycero-phosphate and hexosephosphate. These esters form soluble calcium salts. The normal amount of phosphatase is .15 mg.%. in the plasma. Cartilage has the power to hydrolyse these esters with the deposition of soluble Calcium and Barium Phosphate.

DISTRIBUTION.

In the human adult the richest source is the epithelial portion of the intestinal mucosa. Adult bone contains phosphotases unequally distributed. The enzyme is greatest in the subperiosteal portion

of the bone. Young bone contains more phosphatases than adult bone, the enzyme being greatest in the zone of provisional calcification and in the periosteum.

SIGNIFICANCE OF PLASMA PHOSPHATASE.

Where osteoporosis is active the plasma phosphatase is increased.

In Pagets' disease and osteitis fibrosa diffusa, the amount of the plasma phosphatase may be increased 10 or 20 times.

BONE PHOSPHATASES.

There is little doubt that phosphatases control the deposition of calcium phosphate in growing bones. These enzymes also play a part in the maintenance and repair of bone in adults. Evidence which supports this theory is that phosphatase is present to the greatest amount in growing bone in those areas where deposition of calcium and phosphorus is proceeding rapidly. (26) Recent investigations indicate that Magnesium possesses a specific action on these phosphatase enzymes.

(27) Robinson's hypothesis is that the osteoblasts and hypertrophic cartilage cells secrete an active phosphatase, which, by hydrolising the phosphoric esters of the blood, brings about a local increase in the concentration of phosphoric ions. In

neighbourhood of the affected joint. It is this distinction which differentiates Rheumatoid Arthritis from the other types of arthritis. It is by means of the standard X-ray of the hand that these changes have been noted. However, the main object of this work is to compare the standard hand - using this as an index of calcium changes in the bone - with other diseases where we know that bone calcium changes exist. By this comparison it may help to throw a little light on the etiology of Rheumatoid Arthritis.

THE EFFECT OF PARATHYROID ON THE BLOOD CALCIUM.

The injection of Collip's parathormone will abolish tetany in parathyroidectomised dogs. The administration of this extract to normal dogs raises the blood calcium above normal in proportion to the dose. Greenwald and Gross have demonstrated that after parathyroidectomy of dogs there is a decrease in the excretion of calcium. Parathormone definitely increases the negative calcium balance. The injection of parathormone increases the blood calcium by withdrawing calcium from the bones and increases the deposit of calcium, particularly in the urine. Thus there is a total loss of calcium to the body. It was found that hypocalcalmia was always associated with an increased output. Thus we are justified in

inferring that parathormone given during a deficient calcium intake, causes increased excretion of calcium salts from the bones.

The study of cases of hyperparathroidism due to parathyroid tumours has helped to illustrate the physiology of this gland in relation to calcium metabolism.(28) The excess of activity of this gland leads to a drainage of calcium from the bones, from which ensues a high blood calcium and an increased urinary excretion of calcium. In course of time the process of decalcification produces in the bones a replacement fibrosis and cystic degeneration, leading to the condition, osteitis fibrosa cystica. This is preceded by an increased excretion of Phosphorus which causes a deficiency of phosphorus in the blood. To compensate for this there is a moblization of calcium from the bones.

DISEASES WHERE CALCIUM CHANGES OCCUR IN THE BONE.

Avitaminosis.	Hypervitaminosis D.
Generalised Osteitis Fibrosa.	Rickets.
Osteo Malacia.	Renal Rickets.
Coeliac Rickets.	Osteo Genesis Imperfecta.
Osteitis deformans.	Ex-ophthalmic Goitre.
Chilblain. (Erythema Pernio.)	Leprosy.
The Arthritides.	

A table of the biochemical changes present in some of these conditions is helpful. It crystalises some of these complicated processes. (see page 57a.)

These skeletal dystrophies in which there is an alteration of Calcium and Phosphorus Metabolism and their relationship to the changes in Arthritis, will now be discussed seriatim.

Changes in Blood, Bones, Kidneys, and Catalytic Agents in Various Diseases Affecting Bone.

Disease	Blood Ca	Blood P	Catalyte	Tetany	Principal Bony Changes	Kidneys.
Osteitis Fibrosa	+	—	Parathormone +	0	Osteoclasis	Calculi
Hyper-Vitaminosis D	+	+	Vitamin D +	0	Excessive calcification, followed by reabsorption.	Calculi
Rickets	—	Normal or —	Vitamin D —	+	Deficient calcification and irregular ossification.	
Osteomalacia	Variable	Variable	Vitamin D —	+	Deficient calcification, spontaneous fractures.	
Renal Rickets	—	+	Phosphatase +	+	Delayed union of epiphyses, stunting, irregular ossification.	Chronic nephritis.
Coeliac Rickets	—	Sometimes —	Vitamin D —	+	As in rickets.	
Osteogenesis imperfecta	Normal	Normal	0	0	(Failure of osteoblasts, spontaneous fractures)	
Osteitis deformans	Normal	Normal	Phosphatase +	0	Increased formation of periosteal bone, osteoclasis of cancellous bone.	

HYPERTHYROIDISM.

In a previous paragraph, I have pointed out the intimate relation that exists between the parathyroid hormones and calcium metabolism. The thyroid also plays a certain part and excess or deficiency of thyroid secretion produces changes in the calcium metabolism and in the bone.

In hyperthyroidism there is an increased excretion of calcium by way of the bowel, and yet there is no increase of calcium in the blood. The thyroid also seems to mobilize the calcium from the bones, as in 50% of cases of exophthalmic goitre osteoporosis of the bone occurs. This osteoporosis is produced by a lacunar resorbtion by osteoclasts. Plumer and Dunlop[29] report that they found radiographic evidence of severe osteoporosis in exophthalmic goitre.

It has been observed, as judged from radiograms, that this calcareous demineralization affects the bony system. Aub, writing in the "Journal of Clinical Investigation"[30] states that a controlled hand of an exophthalmic case showed poverty of calcium in the bone.

HYPERVITAMINOSIS D.

In this condition, which to-day can become of practical account, due to the numerous patent

preparations of vitamins on the market, there is a similarity to hyperparathyroidism. In hypervitaminosis D there is an increased absorption of calcium and phosphorus, hypercalcaemia and increased density of bone. Its action seems to be primarily a liberation of phosphoric acid from the tissues, which attracts calcium from the intestines by its acidity; secondarily, by an induced hypercalcaemia which stimulates the parathyroid.

A patient of mine had been taking large doses of Radiostoleum capsules for some months on account of chilblains, and the standard hand in this case is illustrated. In addition she had received intramuscular injections of Calcium Gluconate, and the combination of these two has produced marked increase of bone calcium.

This probably would not have occurred in the absence of the calcium injections, as she adhered to a diet which was very poor in calcium. If this occurs, reabsorption of bone take place, as the bone is called upon to supply the calcium when the exogenous supply is deficient.

With regard to the clinical use of Vitamin D, there is a very small threshold between the optimum therapeutic dose and the toxic dose.

The production of renal calculi have been reported (31)

after the continued administration of 4 mg. daily of irradiated ergosterol.

OSTEITIS DEFORMANS. (PAGET'S DISEASE).

Here again the Blood Calcium and Phosphorus are normal, but changes are taking place in the bone. The plasma phosphatase is, however, high. There is apposition of new periosteal bone and reasorption of cancellous bone. This process is allied to the changes in the bone in Osteo-Arthritis and in Leprosy. Wherever new bone is found, phosphatases are present in excess. There is a disturbance of bone metabolism involving an imbalance between the parathyroids and the suprarenals: there is excess of parathyroid and deficiency of suprarenal cortex hormone. Using the knowledge we have gained by these biochemical investigations, treatment is being tried for this chronic and hitherto incurable disease. Injections of parathormone are given to mobolize the calcium from the bones, and then Vitamin D is administered in graduated doses to effect normal re-deposit.(32)

OSTEITIS FIBROSA.

This condition is associated with an adenoma of the parathyroid gland. The hypercalcaemia, low blood Phosphorus, and increased urinary excretion of both

these elements have already been discussed. It is the significance of the cyst formation, and the analogous change in osteo-arthritis of the hip that will be considered.

According to Struthers (33), areas of translucency shown on the X-ray films which are really areas of replacement fibrosis in healthy bone, are often erroneously regarded as cysts. There are two other conditions to which the term Osteitis Fibrosa has been applied; one of these is a generalised affection occurring in youths without change in the parathyroid system but presenting irregular calcification and general cyst formation in the bones: the other, is a similar condition but limited to a single bone and present in adults. Is it not possible that some of the divergent views about cyst formation in Osteo-Arthritis can be explained by these cases?

The opinion is gaining ground that Paget's disease of the bone and generalized osteitis fibrosa cystica are one and the same disease. From the radiological point of view all transitory stages common to both disorders have been found in each disease.

LEPROSY.

Murdoch and Hunter (34) published an article in the

Americal Journal of Roentgenology. These workers made an interesting radiological study of the joints in cases of leprosy. In most of their cases they took the hand as an indication of the changes which were occurring in the bones. Here again it was found that pathological changes were observed in the hand although clinically there was no sign that any disease process was at work. Out of 140 cases of leprosy, they found 108 had a radiological pathology of the bones. Out of these cases 50% showed changes in the hands and feet. Very few showed bony changes elsewhere. It was observed that the degree of cutaneous involvement was not a reliable index to the amount of bone pathology. The greatest changes in bone leprosy occurred during the formative period and rapid improvement in one phalynx may be accompanied by destructive changes in the others. It would appear that infection was carried to the bone via the vascular supply and there developed into an acute Leprous Osteitis and Osteomyelitis.

The X-ray films which are demonstrated in this article show a wide and extensive variety of bone atrophy and decalcification. This is evidence that there is a disturbance of bone calcium. Some of the films showed cystic degeneration similar to that which occurs in osteitis Fibrosa Cystica. But, as in Arthritis, this is another example of the same agent at work,

producing calcium changes in the bone and cartilage, with subsequent destruction of joints.

The calcium content of the blood in Leprosy is low[35] and a concurrent rise of serum calcium is associated with clinical improvement.

RICKETS.

An interesting report[36] just published gives support to the use of the hand (wrist joint) in the estimation of progress of healing under treatment.

Serial radiograms were taken at weekly intervals, and the rate of cure was determined.

By this measure a standard of optimal rate of cure was established.

1. Between 2-3 weeks the first healing line of calcification in the osteoid zone appears.
2. Between 4-5 weeks there is organisation of the osteoid line with the diaphysis.
3. Between 9-12 weeks a dense, firm zone of calcification with the diaphysis and a normal gap between the epiphysis and the diaphysis takes place.

Further, the blood calcium estimation gave little information in the case of twins which Dr. Spence quotes: one twin was used as a control, and the other was calciferol treated. At the onset the blood calcium was 9.2 mg. per 100 c.c.m. and 9 mg. respectively. After healing, the blood calcium was 10.8 mg., and in the case of the control at the end of 13 weeks, it was 9.9 mg.

Hyperplasia of the parathyroid glands has been shown to occur in both human and experimental rickets.

At a recent meeting of the British Orthopaedic Association,[37] Mr. Stewart succintly described the factors upon which calcium balance depended.

The three factors are:-

1. Calcium intake (diet and vitamin D. activity.)
2. Parathyroid activity (mobilisation of calcium from the bones.
3. Rate of bone-cell proliferation (local phosphatase activity and hydrogen-ion concentration.)

These three factors were interrelated, but the first was of greatest importance in rickets and osteomalacia, the second in osteitis fibrosa, and the third in Paget's disease.

Thus it will be appreciated that these conditions of bony disorder cannot be labelled as separate diseases. They are not disease entities, but rather three bony syndromes which are a manifestation of the following:- hyperparathroidism, hypoparathroidism, hypovitaminosis, genital and neuro-muscular disorders.

Just as these bone diseases do not breed true, but are dependent upon which factor is predominant, so also does the same hold good in chronic arthritis.

CONCLUSIONS.

(1)

Acute cases of Arthritis give no radiological indication as to their nature and therefore it is obvious that a pure radiological classification is impossible.

In the chronic state, radiology is able to give exact information about the condition of the joint and a classification on this basis is the most rational and accurate method.

From the foregoing conclusions, one is reduced to a compromise. A combination of the two methods of investigation, aetiological and clinically, on the one hand and radiological on the other, will yield the most suitable field for future classification.

The acute cases will have a provieional diagnosis. If the Arthritis clear up and leaves no structural change in the joint, then the provisional diagnosis will remain the final diagnosis. On the other hand, if the joint shows radiological changes later on in the course of the disease, the provisional clinical diagnosis is confirmed or corrected and placed in the exact radiological group.

(2)

It will be apparent from a consideration of this work that where the Arthritis is present in the hands, radiology can classify it into one of five groups, namely, Osteo-Arthritis, Rheumatoid Arthritis, Infective Arthritis, Gout, or a mixed type.

Where the Arthritis affects a major joint, the diagnostic hand can, very often, give the key to the diagnosis. This is particularly valuable in cases of gout.

These five groups are exact, and can be used as a basis for the estimation of treatment. At present, in spite of a working classification in Hospital cases, a large personal factor in diagnosis exists. One physician will label one type of case under a certain group, and another physician will label the same case under a different group. It is obvious that no progress can possibly be made in assessing the value of any treatment until something more exact is found. Radiology supplies this want.

(3)

A standard radiograph of the hand should always be taken in every case of Arthritis. This will often give considerable help in diagnosis by supplying

unexpected information and also, in confirming the diagnosis of any other joint involved.

(4)

It will be seen from the tables of classification that no mention is made of Infective Arthritis in the British Medical Association Report. This group comes under the heading of Secondary Rheumatoid. I have endeavoured to show that true Rheumatoid Arthritis is a disease with metabolic changes, and the close resemblance to Grave's Disease and other conditions associated with Calcium upset, places it in a category by itself. In Rheumatoid Arthritis an obvious focus of sepsis is rarely found. It's close association and resemblance to the allergic diseases has also been shown.

In Infective Arthritis of the focal or metastatic type, some virulent micro-organism, usually the streptococcus, is discovered. The morbid anatomy of Rheumatoid Arthritis and Infective Arthritis is different. In the one, you have a lymphocytic reaction and in the other there is a polymorpho-leucocytic reaction, respectively. Clinically, in Rheumatoid Arthritis, there is a polyarthritis and that invariably arising in the small joints of the hand; in Infective Arthritis, one joint only is usually affected.

The X-ray of the hand supplies the information on

which the differential diagnosis between these two conditions can be made. The appearance of the arthritic joint may be clinically identical, but, in Rheumatoid Arthritis, there is a general bone calcium disturbance while in Infective Arthritis, it is purely local, (i.e.) is limited to the component bones of the affected joint.

(5)

There are many cases of chronic Osteo-Arthritis which are, in reality, cases of chronic gout. Although no acute attack of gout has made one suspect this possibility, there is no doubt that a gouty diathesis often exists undetected. The radiological signs, if present, are conclusive and will enable one to give a more specific treatment.

The classical acute gout and the gross tophaceous type are to-day very uncommon but the more insidious type is of very frequent occurence.

Those cases of chronic osteo-Arthritis where gross destruction of cartilage exists, usually have an underlying gouty diathesis. As true degenerative Arthritis does not produce true bony ankylosis some other factor must co-exist; this other factor is gout, and the destruction is brought about by the deposit of uric acid crystals in the cartilage and periarticular structures.

(6)

The distinguishing radiological features between an established osteo-arthritis and an established Infective Arthritis in a major joint are not as definite as the other types. If a single major joint is affected then the classification will depend upon the X-ray appearances of the hand. Should the standard hand show no typical Osteo Arthritic changes, then the major joint will be classified an as Infective Arthritis. If, on the other hand, thereare osteo-Arthritic changes in the standard hand, then the major joint will be classified as an ~~Infective~~ OSTEO-Arthritis.

(7)

In leprosy, degeneration and regeneration can occur simultaneously in neighbouring joints and, therefore, some local factor must be at work as well as general infection or neuro-trophic disturbance.

(8)

In the past one has assumed that by finding a normal blood calcium all must be well with the calcium metabolism. The mechanism which controls the mineral content of the blood is so delicate and complicated that adjustments are soon made between production, excretion and absorption. Therefore, the blood may be in a state of equilibrium at the

expense of some other tissue.

I have shown that there are many diseases where there is an abnormal condition of the bone calcium and yet the blood calcium gives no indication of this. The standard hand is a much more reliable guide in the detection of general calcium imbalance. It can also be used as a guide to treatment.

B I B L I O G R A P H Y .

1. Ch. Arthritis & Fibrositis. Wyatt. 1933. p.24.

2. Causation & Treatment of Arthritis and Allied Conditions. B.M.J. Report of B.M.A. Committee, June 17th 1933.

3. Arthritis. Dr. Figdor. Lancet, August 15th 1931.

4. Aspects of Rheumatism and Gout. Llewellyn Jones Llewellyn. 1927.

5. Aetiological Factors in Osteo-Arthritis of the Hip-Joint. R.C.Elmslie. B.M.J. 7th January 1933.

6. The Pathologenesis of Gout. Knaggs. p.425.

7. Recent Progress in Medicine and Surgery. Sir John Collie. p.226. 1919-1933.

8. Lancet. December 12th 1931. Howitt.

9. Arthritis and Rheumatoid Conditions. Pemberton. 1929. p.53. p.122.

10. Arch. Surg. Dec: 1928. Watt. Vol.17. 1017-1046.

11. Diagnosis in Joint Disease. Allison and Ghormley. 1931. p.2. p.70. p.137.

12. Rad. Sides of Chronic Arthritis. Alex.Thompson. Proc. Roy. Soc. Med. 1929. p.1119.

13. Chronic (non-tuberculous) Arthritis. Fisher. p.43-44.

14. Recent Advances in the study of Rheumatism. Poynton and Schlesinger. p.201.

15. Macroscopic and Microscopic Morbid Anatomy of Arthritis Deformans. Nichols and Richardson. 1909. Journal Med. Res. Vol.21. p.149.

16. Proc. Roy. Med. Strangeways. 1924. Vol.17. 1. p.12.

17. The Radiographic Dissection of Chronic Rheumatic Arthritis. G. Scott. B.M.J. 13th Feb: 1932.

18. Discussion on Arthritis at Centenary Meeting. B.M.A. Scott.

19. Recent Advances in Radiology. Peter Kerley. 1931. p.40.

20. Chronic (non-tuberculous) Arthritis. Fisher. p.107.

21. The Radiology of Bones and Joints. Brailsford 1934. p.24. p.446.

22. Peut-on diagnostiquer La Goute au moyen de la Radiographie? Weil et Détré. La Presse Medicale. 18th March 1925. p.351-353.

23. Hypodermolithiasis. Patterson. B.M.J. 10th February 1934.

24. The Significance to Clinical Medicine of Studies in Calcium and Phosphorus Metabolism. Ext. from Goulstonian Lectures in 1930. Hunter. Pub. Lancet. April 26th 1930.

25. Clinical Interpretation of Aids to Diagnosis. Daniel T. Davies. Vol.2. p.92.

26. The Mineral Basis of Life. Sheldon. B.M.J. 13th January 1934. p.47.

27. Phosphorus Metabolism. R. Robinson. Biochemical Journal 1923. XVII. p.286. and 1926 XX. p.388.

28. Ca. Metabolism and the Parathyroids. Langdon Brown. Pot. Grad. Medical Journal. Nov: 1932.

29. Proc. Staff Meetings. Mayo Clinic 1928. Plummer and Dunlop. III. 119.

30. Journal of Clinical Investigation. Aub. 1929. VII. p.97.

31. Endocrinology; Recent Progress in Med. and Surgery. Langdon Brown. p.17.

32. How do Drugs Act? Langdon Brown. B.M.J. 2nd December 1933.

33. Parathyroid Osteodystrophy. Struthers. Clinical Journal. Oct: 1933. p.429-430.

34. Leprosy: a Roentgenological Survey. Murdock and Hutter. The American Journal of Roentgenology and Radium Therapy. Vol. XXVIII. No. 5. Nov: 1932.

35. Treatment of Leprosy by means of Alepol Tablets. Leprosy Rev: Oct.1933. Badenock. p.144-146.

36. Antirachitic Activity of Calciferol. Spence. Lancet. Oct: 21st. p.911. 1933.

37. Meeting of the British Orthopaedic Association in Edinburgh. Nov: 24th and 25th 1933. C.R. Stewart.

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The following tables represent a series of cases which I investigated at the British Red Cross Clinic, at Marylebone. These cases were picked out specially from a large amount of material to enable me to illustrate the main features of my Thesis.

NAME	OCCUPATION	AGE	SEX	PATIENT'S COMPLAINT AND DURATION.
Freeman. (S)	Nurse.	18	F	Pain in right hip and left shoulder. 1 year.
Parker. (M)	Station master.	61	M	Pain in right arm, right leg and back of neck. 4 - 5 years.
White. (M)	Baker.	49	M	Pain in left hip, right elbow and right wrist. Nine years on and off.
Grigg. (M)	House-keeper.	33	F	Rheumatism in left ankle. Began four years ago.
Barnes. (S)	Maid	54	F	Pain and stiffness in both knees. 1 year.
Woods. (M)	Dairy-keeper.	59	F	Pain in right hand, shoulder, hip and foot. Three years on and off.
Smith. (M)	House-keeper.	48	F	Pain and stiffness in left thigh. 2 years.

CLINICAL DIAGNOSIS.	X-RAY HAND.	X-RAY OTHER JOINTS.	SPECIAL FEATURES.
Fibrositis.	No change.	No change.	
Unclassified Arthritis. ? Infective.	No change.	Right knee - osteo-arthritic type.	Rheumatic fever at 13 and 29 years.
Unclassified Arthritis.	Right hand - infective arthritis, bony ankylosis.	Left sacro-iliac normal. Left hip infective arthritis; loss of cartilage; irregularity of articular surface.	Pyorrhoea.
Rheumatoid Arthritis.	Ankylosis mainly fibrous, except little finger. Atrophic arthritis. (advanced.)		
Osteo-arthritic knees.	Right hand - no change.		Rheumatic fever at 15 years. T.B. left lung at 17 & 21 years.
Unclassified Arthritis.	Osteo-arthritic changes in several joints.	Right hip - no change. Some sclerosis of right sacro-iliac joint; osteophytic lipping of lower lumbar spine.	Hysterectomy at 30 years. Heberden's Nodes.
Osteo-Arthritis.	Osteophytic formation especially at metacarpal phalangeal joint.	Left hip - complete loss of cartilage. Sclerosis of head and acetabulum; cavitation; osteo-arthritic group. (Infective).	2-3 miscarriages. Gums - streptococci haemolyticus, staphylococci. Vagina streptococci & staphylococci.

NAME	OCCUPATION	AGE	SEX	PATIENT'S COMPLAINT AND DURATION.
Burrows. (M)	House-keeper.	43	F	Pain and stiffness in back, elbows and knees. 2 years.
Higginson. (S)	Clerk.	69	F	Pain in thigh going down to to left knee. Hand and wrist. 5 years.
Thacker. (M)	House-keeper.	59	F	Pain and stiffness in different joints. 20 years.
Thomson. (S)	Clerk.	49	F	Swelling and aching, both knees. 5 years.
Burrowes. (S)	---	56	F	Painful elbow and stiffness other joints. 18-19 years.

CLINICAL DIAGNOSIS.	X-RAY HAND.	X-RAY OTHER JOINTS.	SPECIAL FEATURES.
Rheumatoid Arthritis.	Rheumatoid arthritis advanced: loss of cartilage of wrist and minor joints.	Right knee - bone decalcification: loss of cartilage under patella.	Teeth extracted with no benefit. Menstruation irregular.
Gout. ? osteo-arthritis.	1. osteoporosis.(Senile) 2. Fusion of carpals. 3. Destruction of cartilage. 4. Sclerosis of bones of wrist.	Left knee - osteoporosis (Senile). Destruction of cartilage. In addition, there is lateral displacement of femur inwards and new bone formation. Infective arthritis.	Bronchitis and pleurisy. Menopause -52 Blood uric acid 3.6 mg.%. Faeces - staphylococci, B. Coli.
Degenerative Arthritis.	Osteo-arthritic changes, especially in metacarpal and phalangeal joints.	Lumbar, spinal and sacro-iliac joints. No change.	In 1930 Tonsilitis.
Metabolic arthritis.	Osteophytic formation. periarticular ossicles.	Osteophytes - patella.	1925 - Hysterectomy.
Senile Arthritis.	General decalcification (senile) and slight osteo-arthritic change.	Right hip, very definite decalcification of bone (senile). No definite loss of cartilage. Osteophytic formation. Spine appears to be ankylosed.	Late onset of menstruation. Early menopause.

NAME	OCCUPATION	AGE	SEX	PATIENT'S COMPLAINT AND DURATION.
Gee. (M)	---	64	M	Pain right knee, both shoulders, stiffness and limitation of movement. 3 years.
Bailey. (M)	Grocer.	62	M	Pain in knees, ankles, shoulders and hands. 8 years.
Venner. (S)	Chimney sweep.	24	M	Pain in feet and knees. 2 months.
Pipe. (M)	Grocer.	62	M	Pain in feet and hands. 2 years.
Thomas. (M)	House-keeper.	47	F	Stiffness and pain, generalized: 10 years.
Hillditch. (M)	Clerk.	33	F	Pains in hands and fingers: On and off, 2 years.

CLINICAL. DIAGNOSIS.	X-RAY HAND.	X-RAY OTHER JOINTS.	SPECIAL FEATURES.
Osteo-Arthritis.	No change.	Right knee - nil. Left hip, osteo-arthritic changes; loss of cartilage; osteophytic formation; atheromatons arteries.	Bacteriology - Throat - micro-coccus catarr-halis. Throat and teeth - streptococcus longus. Urine - strepto-coccus haemoly-ticus and staphy-lococci. Faeces - Bacillus Coli.
Osteo Arthritis.	No change.	Left foot and left knee. No change.	1926 - Teeth extracted: thought to be the cause.
Fibrositis.	No change.	No change.	
Infective Arthritis, unknown cause.	No change.	Right foot - flat.	
? Gout.	No change.	Sacro-iliac - normal joint.	
? Early Arthritis.	Right hand. No gross change.	- - -	Patient cleared quickly. X-ray of use.

NAME	OCCUPATION	AGE	SEX	PATIENT'S COMPLAINT AND DURATION.
Smith. ( )	Provision Manager.	43	M	Pain in neck, head and hands. 9 years.
Manuel. (M)	Clerk.	30	M	Grating and pain left knee. 3-4 months.
Woodham. (M)	House-keeper.	52	F	Stiffness right leg and knee. 8 years ago.
Ford. (M)	Nil.	54	F	Rheumatism all over. 5 years ago.
Warwick. (S)	Clerk.	54	F	Polyarticular stiffness and pain in knee, ankles, hand and wrist. 5 years.
Higo (S)	Companion.	48	F	Pain and stiffness. Knees, feet, ankles, shoulder, wrists and hands. 2 years.

CLINICAL DIAGNOSIS.	X-RAY HAND.	X-RAY OTHER JOINTS.	SPECIAL FEATURES.
Infective Arthritis.	Right - loss of cartilage. Area of rarefaction. Alteration of bone texture.	Chest - no gross change. Difficult to classify. Probably unusual form of infective arthritis.	4 years ago X-ray of knee. Said to have arthritis. Operation, arthrodesis of right knee joint for ? T.B. Previous illnesses- pleurisy, pneumonia.
Fibrositis.	Nil.	Patella - old injury cartilage.	Enlarged Tonsils and recurrent Tonsilitis.
Senile osteo-arthritis.	Nil.	Nil.	
Unclassified. ? menopausal arthritis.	—	Left ankle - rheumatoid arthritic change and loss of cartilage - all joints.	Bacteriology. Urine - staphylococci. Gum cultures: diphtheroids and staphylococci.
Gouty Arthritis.	Loss of cartilage; destruction of articular ends of bones in many minor joints. Fusion of carpals. Rheumatoid arthritis advanced.	Right knee - Excess of bone upper end of tibia. Articular surface.	Hand condition preceded by chilblains. Blood uric acid 2.7 mg.%.
Rheumatoid Arthritis following shock.	Early fusion of carpals. Decalcification: no loss of cartilage.	Right knee - Osteoma. An exostosis growing from tuberosity of tibia. Loss of cartilage. Rheumatism not very active.	Menopause - 3 weeks after onset of arthritis.

NAME	OCCUPATION	AGE	SEX	PATIENT'S COMPLAINT AND DURATION.
Clarke. (M)	Carpenter	69	M	Pain and stiffness, arm and leg. 12 years.
Kirby. (M)	House-keeper.	58	F	Pains in knee and hip. Getting worse. 2 years.
Parkes. (S)	Missionary	51	F	Pain in back - 4 years. Pain in shoulder 9/12 yr.
Lucas. (S)	Domestic	26	F	Pain and swelling of hands, knees and feet. 3½ years.

CLINICAL DIAGNOSIS.	X-RAY HAND.	X-RAY OTHER JOINTS.	SPECIAL FEATURES.
Myositis and Neuritis.	Decalcification (Senile).	Hip - Infective type of osteo-arthritis. 1. loss of cartilage. 2. Decalcification. 3. Slight cavitation. 4. Osteophytes.	Bacteriology - Urine - B.Coli and diptheroids Haemolyticus Streptococcus. Throat - streptococcus longus. M. catarrhalis. Faeces - B.Coli and diptheroids.
Unclassified.	Right hand - general decalcification; fusion of carpal and multiple joint involvement.	Right knee - Some cartilage loss especially under patella. Roughening of joint surfaces; overgrowth of bone. In spite of excess of bone in knee, the changes are rheumatoid.	Limitation of movement of both wrists. Crepitus shoulder joint with limitation of movement. Ditto - knees.
Rheumatoid Arthritis.	Right hand -loss of cartilage. Rheumatoid arthritic group. (3 months later) no change.	Cervical and dorsal vertebrae, no change. 3 months later - osteophytic formation -(D10 - L3). Sacro-iliac - partial ankylosis; ligamentous ossification between lumbar vertebrae; general calcification.	1927 - Cholecystectomy. Bacteriology - Urine - staphylococci and streptococci. Faeces - B.Coli.
Arthritis of Rheumatoid type.	Right & left hands; (interesting,) wrists and several minor joints show infective type of arthritis - in addition, the infection has involved the periosteum of several long bones, leading to a deposit of new bone. Loss of cartilage of wrists and several minor joints. No disturbance of bone calcium. Long exostosis from lower end of right femur.	- - - -	Bacteriology - Vagina: - staphylococci aureus and diphtheroids. Naso-pharynx - micrococci catarrhalis and streptococci. Faeces - B.Coli. Urine - staphylococci albus and diphtheroids.

R H E U M A T O I D

NAME & OCCUPATION.	SEX	AGE	CLINICAL DIAGNOSIS AND DURATION.	JOINTS INVOLVED.
Ford, (M) Nil.	F	55	Menopausal arthritis. 7 years.	Hands and ankles.
Honrow, (M) Nil.	F	51	Infective arthritis. ?	Elbows, wrists and knees.
Dann, (W) Boarding-house.	F	37	Infective arthritis. 7 years.	Hands, elbows and knees.
Philpott. ( ) Housekeeper.	F	49	Rheumatoid arthritis. 2 years.	Multiple joint involvement.
Fawcett, (S) Teacher.	F	54	Rheumatoid arthritis. 2 years.	Shoulder, elbow, wrist, finger and knees.
Speidel, (W) Housekeeper.	F	56	Rheumatoid Arthritis.	Hands and feet cervical, spinal.
Winder, (S) Palourmaid.	F	48	Menopausal arthritis.	Hands, shoulders, feet and knees.
Bullen. ( ) Housekeeper.	F	29	Fibrositis. 5 months.	Shoulder, lumbar regions.

A R T H R I T I S .

X-RAY OF HAND.	X-RAY OF OTHER JOINTS.	SPECIAL FEATURES.
Rheumatoid.	Rheumatoid.	
Rheumatoid Group.		Very acute case - in bed 5 weeks, 1932.
Rheumatoid Group.	Loss of cartilage of major and minor joints, with general osteo-porosis.	
Rheumatoid Group.		
Left hand - marked decalcification of bone. Rheumatoid Group.	Right elbow. No gross changes.	
Typical Rheumatoid Arhtritis. Active 3rd. degree.	Cervical spinal. No bone or joint changes.	
Rheumatoid Arthritic group.		
Atrophic group.	Hip - head of femur flattened and pro-jects out of socket more than it should. Arthritis secondary.	